THE ROCKEFELLER INSTITUTE
FOR MEDICAL RESEARCH
66TH STREET AND YORK AVENUE
NEW YORK 21, N. Y.

November 21, 1952

Dear Josh:

As a consequence of the postulated segregational lag in transduction the transinductions should not be clonal, and I thought it of some import to study this in another way, and if it were so when and how long should also be answered. The preliminary experiments verify the conclusion. They were set up as follows: At time zero a number of EMB gal and NSA plates were spread with a known number of bacteria and phage (gal+, S^r FA). The rest is obvious, respreading some plates after intervals of incubation with streptomycin as necessary and washing others for growth rates. The gave five and one-half generations observed a generation time of 37 minutes including the initial lag. In order for any differences in growth rate between phage infected and non-infected bacteria to be equilibrated out, the initial multiplicity was about two. At about five generations the number of transinductions doubles while prior to that it is pretty level considering the large variance to be expected. With streptomycin a surprising thing happens, the number of zero points is almost as high as those after one generation. This means that there must be a long delay in SM action which is both advantageous and disadvantageous. We can't study the phenomic lag but then again phenomic lag doesn't much enter into our scoring of the repositioned transinductions.. A good unrelated phage resistance marker would help. Do you think that replica plating is efficient enough to do further work with that procedure, or is it only useful to say that something is clonal and not vice versa? Of course reconstruction experiments must be done to show that growth rates are equivalent and the lowering of the growth rate of the cell in the process of transductior ruled out by use of sufficient number of characters.

Best regards

Sincerely,

Norton